CASE REPORTS

Refer to: Brant-Zawadzki M, Wofsy CB, Schechter G: CT evidence of subarachnoid hemorrhage due to presumed gnathostomiasis. West J Med 1982 Jul; 137:65-67

CT Evidence of Subarachnoid Hemorrhage Due to Presumed Gnathostomiasis

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SPONTANEOUS SUBARACHNOID HEMORRHAGE is due to aneurysmal rupture in most cases and is easily detected with computerized tomography (CT). Nevertheless, approximately 20 percent of cases of subarachnoid hemorrhage documented with lumbar puncture have no explainable cause on subsequent diagnostic evaluation. Parasitic infestation with *Gnathostoma spinigerum* is a surprisingly frequent cause of subarachnoid hemorrhage in the pediatric and adolescent populations of Indochina.[1,2] The presence of a meningomyeloencephalitis with cerebrospinal fluid (CSF) eosinophilia is a strong clue to the cause in such cases. *Angiostrongylus cantonensis,* another nematode found in the Far East, can produce similar features but has a lesser predilection for the spinal cord and generally causes a less severe infection.[3] In view of the recent influx of Indochinese immigrants to this country, it is important for clinicians to be aware of these entities. We report a case of a patient in whom meningomyeloencephalitis with CSF eosinophilia developed and who suffered a subarachnoid hemorrhage on his arrival in the United States.

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Report of a Case

A 21-year-old Laotian man was in transit to the United States from Thailand when he noticed weakness of both legs. On arrival in San Francisco, he was too weak to deplane and was admitted to the US Public Health Service Hospital. He had symptoms of headache, weakness in both lower extremities and inability to urinate. The history was remarkable for leprosy, diagnosed and treated in Laos. Physicial examination on admission showed normal vital signs and no abnormalities. Neurologic examination showed a normal mental status and profound weakness of both lower extremities with areflexia. Sphincter tone was diminished and Babinski's reflex was positive bilaterally.

Laboratory data showed an eosinophilia (22 percent) with a normal leukocyte count. Analysis of the CSF specimen obtained by lumbar puncture on the second day revealed a glucose level of 24 and a protein level of 228 mg per dl, with 1,200 leukocytes (62 polymorphonuclear cells, 29 eosinophils and 8 lymphocytes). Peripheral nerve conduction studies suggested a proximal degeneration of the plexus root or anterior horn cells. Studies of stool specimens for ova and parasites showed *Clonorchis sinensis, Entamoeba coli,* hookworm and *Strongyloides stercoralis.* VDRL was negative. Thiabendazole therapy was instituted.

During his initial hospital course, a sudden right-sided headache and emesis developed with neck stiffness and facial weakness, followed closely by an altered sensorium. The patient was transferred to San Francisco General Hospital for myelography and CT scanning. He remained afebrile. He again was noted to be profoundly weak, with ankle clonus and absent patellar reflexes. Headache, neck stiffness and lethargy persisted.

Myelogram showed no obvious abnormality. The CT scan showed evidence of subarachnoid blood in the perimesencephalic cistern and fourth ventricle (Figure 1). Lumbar punctures again showed CSF evidence of inflammation, with eosinophilia, protein levels in the 100 to 228 mg per dl range, red cells and xanthochromia. Repeated cultures of a CSF specimen for bacteria, acid-fast bacilli, fungi and viruses were negative. Viral

serologies for poliomyelitis, echovirus, coxsackievirus and influenza were negative. Repeat ova and parasite studies of stool specimens revealed further infection with *Taenia saginata* and *Opisthorchis viverrini*. Mebendazole therapy was instituted.

The patient gradually improved, slowly regaining strength in his lower extremities. Follow-up CT scan showed resolution of the subarachnoid hemorrhage. Specimens of serum and CSF, which were sent to the Centers for Disease Control (CDC) in Atlanta to be examined for serologic evidence of *A cantonensis* infestation, were negative.

Discussion

G spinigerum infestation is widespread throughout Asia. Inadequately cooked food, which harbors the third-stage larvae, is the mode of transmission of the agent to humans. The parasite migrates easily through any soft tissue, including that of the central nervous system. It causes damage to neural tissue and blood vessels because of its excessive activity and spinous body. Radiculomyelitis, radiculoencephalomyelitis and subarachnoid hemorrhage are the common manifestations of central nervous system (CNS) *Gnathostoma* infection. Profound CNS involvement and eosinophilia in CSF specimens are initial clues to the diagnosis. Recovery of the parasite from tissue provides definitive diagnosis.[1-4]

Our patient initially suffered symptoms of a myelitis that then progressed to a more generalized encephalomyelitis. Eosinophilia in peripheral blood and CSF specimens was recorded. Clinical and CT findings supported a diagnosis of subarachnoid hemorrhage. In short, the presentation was typical of gnathostomiasis.[3] No other parasites found in this patient could have produced the symptom complex. No other infectious or vascular cause was reported despite extensive examination. Because tissue specimens (usually obtained at autopsy) are needed to definitely establish the diagnosis of gnathostomiasis, only a strongly presumptive diagnosis can be made in this case. The other parasitic infestation that produces CSF eosinophilia and diffuse CNS involvement is *Angiostrongylus,* for which there was no serologic evidence in this case. Although CSF eosinophilia is common in meningoencephalitis due to *Angiostrongylus,* subarachnoid bleeding is

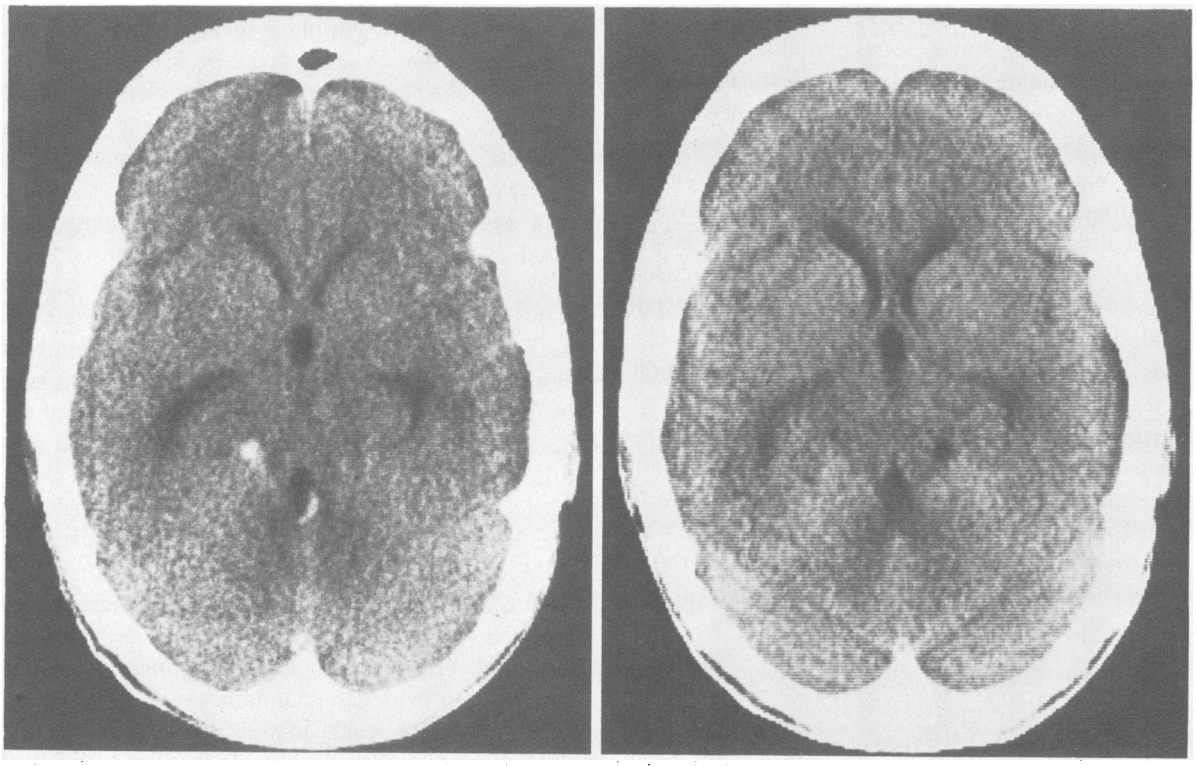

Figure 1—Computerized tomography (CT) scan of 21-year-old Laotian man with gnathostomiasis shows presence of subarachnoid blood in the right ambient cistern and roof of fourth ventricle eight days after the onset of symptoms **(left).** CT scan done one week later shows the clearing of blood from these spaces **(right).**

rare and the tendency for spinal cord involvement much less prominent.[3,4]

CT scanning is quite sensitive for subarachnoid bleeding in the intracranial space. Patients with aneurysmal subarachnoid bleeding generally have hemorrhage detected in the basal cisterns.[5,6] The atypical location of subarachnoid blood on CT scans, in the setting of a Southeast Asian with typical diffuse CNS signs and evidence of inflammation with eosinophilia in the cerebral spinal fluid, should raise the question of *Angiostrongylus* or *Gnathostoma* infestation; the presence of myelitis favors the latter.

REFERENCES

1. Visudhiphan P, Chiemchanya S, Sonburanasin R, et al: Causes of spontaneous subarachnoid hemorrhage in Thai infants and children—A study of 56 patients. J Neurosurg 1980 Aug; 53: 185-187

2. Boongird P, Phuapradit P, Siridej N, et al: Neurological manifestations of gnathostomiasis. J Neurol Sci 1977 Mar; 31(2): 279-291

3. Punyagupta S, Juttijudata P, Bunnag T: Eosinophilic meningitis in Thailand—Clinical studies of 484 typical cases probably caused by *Angiostrongylus cantonensis*. Am J Trop Med Hyg 1975 Nov; 24(6 Pt 1):921-931

4. Kuberski T: Eosinophils in the cerebrospinal fluid. Ann Intern Med 1979 Jul; 91:70-75

5. Davis JM, Davis KR, Crowell RM: Subarachnoid hemorrhage secondary to ruptured intracranial aneurysm: Prognostic significance of cranial CT. AJR 1980 Apr; 134:711-715

6. van Gijn J, van Dongen KJ: Computerized tomography in subarachnoid hemorrhage: Difference between patients with and without an aneurysm on angiography. Neurology 1980 May; 30: 538-539

Refer to: Valenta LJ, Eisenberg H, Miller DR, et al: The value of artificial beta cell in the management of insulinoma. West J Med 1982 Jul; 137:67-75

The Value of Artificial Beta Cell in the Management of Insulinoma

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INSULINOMA, a tumor of the pancreatic beta cell causing hypoglycemia, presents a number of diagnostic and therapeutic problems. When use of exogenous insulin can be excluded, a combination of hypoglycemia and inappropriately high serum insulin levels (above 6 to 10 μU per ml) is diagnostic.[1] However, a number of insulinoma patients have fasting euglycemia.

Insulinomas are usually small and difficult to see by radiographic techniques. Even inspection and palpation of the pancreas during surgical procedure may fail to locate the tumor. Occult and multiple lesions are often difficult to find at operation and may require major pancreatic resection, sometimes without relief of symptoms.[2]

This case demonstrates the difficulties of clinical and laboratory diagnosis in a patient with insulinoma, the diagnostic value of subselective angiography and the use of the artificial beta cell in the management of patients.

Report of a Case

The patient is a 26-year-old woman whose principal complaint was periodic loss of consciousness. The first episode occurred at the age of 12 and was preceded by symptoms of catecholamine excess. Later on, episodes of loss of consciousness occurred about every other month and were then introduced by a prodromal period of depersonalization, drowsiness and sometimes generalized jerking movements. The patient said she had no feeling of increased hunger, restlessness, blurred vision, palpitations or any gastrointestinal symptoms. Repeated brain scans and electroencephalograms showed no abnormalities. Nevertheless, a diagnosis of a seizure disorder was made and the patient was started on a regimen of diphenylhydantoin (phenytoin; Dilantin) and phenobarbital, which she had been taking since. There was no relief of the symptoms, and the dosages of phenobarbital and Dilantin were increased progressively. Over the years the frequency of the

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